

Anesthetic and postanesthetic management of sea turtles

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Objective—To examine the physiologic effects of inhalation anesthesia in aquatic turtles to improve anesthetic techniques and postanesthetic monitoring.

Design—Retrospective case series.

Animals—9 Kemp's ridley sea turtles.

Procedure—Isoflurane was used as the general anesthetic during 14 minor surgical procedures. Turtles were orotracheally intubated, and a surgical plane of anesthesia was maintained with $2.7 \pm 0.4\%$ (mean \pm SE) isoflurane. The duration of anesthesia was 131 ± 12 minutes. Pulse rate, blood pressure, blood gases (PaO_2 and PaCO_2) and pH, blood lactic acid concentration, and capnography were used to evaluate the physiologic responses of sea turtles to isoflurane.

Results—An isoflurane concentration of $3.4 \pm 0.3\%$ provided anesthetic induction in 7 ± 1 minutes. The mean duration of the recovery phase was 241 ± 31 minutes. The duration of the recovery phase was not affected by the duration of anesthesia, type of carrier gas, method of ventilatory weaning, or use of selected pharmacologic agents. The recovery phase was characterized by hypoxemia, progressive acidemia, hypercapnia, and lactic acidosis. Awakening in the turtles was preceded by a characteristic tachycardia and tachypnea. All sea turtles recovered from isoflurane anesthesia without apparent adverse effects within 24 hours.

Clinical Implications—Isoflurane appears to be safe and effective in providing surgical anesthesia in turtles that require a timely return to an aquatic environment. This study should assist veterinarians in predicting the physiologic responses of aquatic turtles to inhalation agents. (*J Am Vet Med Assoc* 1996;208:720-726)

Injectable agents administered by IV, IM, or IP routes are widely used for general anesthesia of reptiles.¹ Typically, IV injection in many reptile species is limited by lack of an appropriate, easily accessible venous site. Sea turtles, however, possess a dorsal cervical sinus that is readily available for blood sampling² and injection of anesthetic agents.³ Nevertheless, potential

variation in drug sensitivity may exist between species. For example, there was unacceptable morbidity and mortality when we attempted to anesthetize Kemp's ridley sea turtles (*Lepidochelys kempi*) by administering pentobarbital sodium into the dorsal cervical sinus, as has been described for green sea turtles (*Chelonia mydas*).³

Butler et al⁴ reported that green sea turtles anesthetized with 2 to 3% halothane recovered from anesthesia within 5 to 60 minutes, whereas recovery time in the same species given 10 to 25 mg of pentobarbital sodium/kg of body weight, IV, was 4 to 24 hours.³ More recently, Shaw et al⁵ reported that green and loggerhead (*Caretta caretta*) sea turtles anesthetized with isoflurane recovered within 2 to 6 hours. These data suggest that use of inhalant anesthetic agents may reduce the duration of the recovery phase and, therefore, might be more appropriate for reptile species that require timely postoperative return to an aquatic environment.

The purpose of the study reported here was to examine the physiologic effects of 14 anesthetic procedures using isoflurane in 9 Kemp's ridley sea turtles to determine whether improvements in anesthetic techniques and postanesthetic monitoring could be made in aquatic turtles.

Materials and Methods

The turtles used were part of a group of captive-reared Kemp's ridley sea turtles housed and maintained at the National Marine Fisheries Service, Galveston, Tex, as previously described.⁶ The 9 turtles used were unreleasable into their natural habitat as a result of congenital deformities (eg, reduced flipper length). Food was withheld from the turtles approximately 12 hours prior to surgery, and the turtles were transported to the research facility on the day of surgery. Preoperative weight, pulse and respiratory rates, and cloacal temperature were measured and recorded. Prior to surgery, the head, neck, and carapace were scrubbed lightly to remove adhering materials, such as algae and barnacles.

Turtles were orotracheally intubated.⁷ Briefly, 2 polyvinylchloride rods were used as mouth gags to permit inspection and manual opening of the glottis with the tip of the endotracheal tube. Intubations often were difficult, because the laryngeal sphincter muscles remained tightly closed. One turtle was given succinylcholine chloride (0.5 mg/kg, IM) to facilitate intubation. The succinylcholine had no observable effect at this dosage and was not used on any other turtle. Anesthesia was induced by administering 2 to 5% isoflurane^a in 2 L of carrier gas/min through a semiclosed rebreathing anesthesia circuit connected to the endotracheal tube. The carrier gas was 100% O₂, 5% CO₂/95% O₂, or 21 to 40% O₂ (balance N₂). The N₂-O₂ mixtures were produced by use of an air-O₂ blender.^b Composition of all inspired and expired gases was monitored by use of raman gas spectrography^c (equipment was manually calibrated prior to each study with known concentrations of isoflurane, CO₂, and N₂). Turtles were mechanically ventilated by hand until their general

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movement ceased and their palpebral reflex slowed. At this time, 1 polyvinylchloride rod was removed, and the endotracheal tube was inserted through a hole in the second rod. This protected the endotracheal tube and prevented airway occlusion. Surgical anesthesia was maintained, using a vaporizer setting of 0.5 to 4.3% isoflurane in 1 L of carrier gas/min.

Ventilation was supported throughout surgery by continued hand ventilation or, if the estimated tidal volume was ≥ 100 ml, by a volume-limited, time-cycled mechanical ventilator^d at a rate of 2 to 8 breaths/min. Inspiratory flow was adjusted to 1.5 to 1.7 seconds inspiratory time. The inspiratory time was determined from comparable data for the green sea turtle,⁴ as well as from estimations of inspiratory time in awake, spontaneously breathing Kemp's ridley sea turtles. Standard mammalian formulas were not accurate for estimating tidal volume in these reptiles, because the relationship between lung volume and body weight is not the same in the 2 phyla.⁸ Initially, tidal volumes of 23 to 31 ml/kg were used, on the basis of reports for the loggerhead sea turtle.⁹ Tidal volumes were determined later to be adequate when the front flippers moved outward slightly during inspiration. The delivered tidal volumes were measured from the bellows setting on the ventilator, and the resultant peak inspiratory pressure was recorded from the pressure gauge located on the inspiratory limb of the anesthetic circuit. Tidal volumes and inspiratory pressures in this study were estimations of the actual values, because neither piece of equipment was calibrated.

Turtles were placed in dorsal recumbency and given lactated Ringer's solution (5 ml/kg, IP). Turtles also received chloramphenicol sodium succinate (70 mg/kg, IM) or enrofloxacin (2.5 mg/kg, IM) prior to surgery. The surgical site was aseptically prepared for occlusive cannulation of the right carotid artery with polyethylene tubing. A second surgery was performed 7 to 10 days later to remove the catheter from 5 of the 9 turtles.

Pulse rate was monitored throughout the study, using an ultrasonic Doppler flow probe^e placed over the femoral triangle, thoracic inlet, caudodorsal aspect of the front flipper, or dorsal cervical sinus. Heart rate was unreliably monitored via electrocardiography (lead-II), by attaching the leads to the flippers or the axillary and femoral regions with clips, needles, or pregelled, self-adhering electrode pads. An esophageal stethoscope was used to try to monitor heart rate in 3 turtles; however, keratinous esophageal protrusions prevented placement of the stethoscope past the pharyngeal region. A pulse oximeter probe^f attached to various flipper sites was ineffective in obtaining a quality signal, possibly because of poor pulsatile blood flow in the flipper regions. Cloacal temperatures, which were measured with a thermistor,^g were maintained at 27 to 29 C during surgery and at 24 to 30 C during recovery by use of a circulating warm-water pad under the turtle and an overhead heat lamp.

Arterial blood pressure tracings were obtained during and after surgery by connecting the carotid catheter directly to high-pressure extension tubing and a pressure transducer. The pressure transducer was zeroed at the level of the thoracic inlet and was calibrated prior to each study with a mercury manometer. Systolic (SBp) and diastolic (DBp) blood pressures and pulse rate were continuously recorded on a multichannel chart recorder.^h

Blood samples were collected during and after surgery from the carotid artery catheter of 3 turtles with heparinized glass syringes to measure arterial blood gases (P_{aO_2} and P_{aCO_2}), pH, and lactic acid concentration. The total blood sampling volume was $< 1\%$ of body weight. Samples were immediately placed on ice and were measured within 1 hour of collection, using individual pH and gas electrodesⁱ ther-

Table 1—Summary of physiologic data and anesthetic management for 9 Kemp's ridley sea turtles undergoing 14 surgical procedures

Period	Variable	Mean \pm SE	Range	N
Preoperative	Pulse rate, beats/min	34 \pm 3	14–50	13
	Respiratory rate, breaths/min	2 \pm 1	0–3	5
	Cloacal temperature, C	25 \pm 3	22–27	2
Intraoperative	Induction, % concentration of isoflurane	3.4 \pm 0.3	1.5–5.0	14
	Maintenance, % concentration of isoflurane	2.7 \pm 0.4	0.5–4.3	14
	End-tidal, % concentration of isoflurane	2.0 \pm 0.3	1.1–3.7	6
	End-tidal CO_2 , mm of Hg	14 \pm 1	12–18	6
	Tidal volume, ml/kg	10 \pm 1	7–14	7
	Inspiratory pressure, cm of H_2O	15 \pm 1	10–20	13
	Pulse rate, beats/min	15 \pm 1	4–32	13
	Systolic pressure, mm of Hg	31 \pm 6	17–44	4
	Diastolic pressure, mm of Hg	20 \pm 4	13–26	4
	Respiratory rate, breaths/min	3.7 \pm 0.4	2–8	14
Recovery	Pulse rate, beats/min	15 \pm 1	4–39	14
	Systolic pressure, mm of Hg	40 \pm 4	30–50	6
	Diastolic pressure, mm of Hg	25 \pm 3	14–33	6
Awake	Pulse rate, beats/min	50 \pm 2	38–60	13
	Systolic pressure, mm of Hg	46 \pm 4	34–59	5
	Diastolic pressure, mm of Hg	39 \pm 2	33–44	5
	Respiratory rate, breaths/min	8 \pm 1	4–12	13

mostated to cloacal temperature. Blood concentration of HCO_3^- was calculated from a rearrangement of the Henderson-Hasselbalch equation, using values for αCO_2 and pKa derived for the plasma of Kemp's ridley sea turtles.¹⁰ Lactic acid concentration was determined enzymatically.^j Results of preliminary experiments indicated that storage of blood samples for 1 hour did not result in changes in pH, PO_2 , PCO_2 , and lactic acid concentration.

After termination of anesthesia, the breathing circuit was flushed with 100% O_2 to decrease the inspired isoflurane concentration to $< 0.1\%$. Then, we attempted to reduce the duration of the recovery phase via several experimental manipulations. First, the composition of inspired gases was altered during anesthesia and recovery. Four turtles inspiring 100% O_2 during anesthesia continued to inspire 100% O_2 during recovery. In 3 other turtles, the inspired gas was changed from 100% O_2 during anesthesia to 21% O_2 (balance N_2) during recovery. Six turtles receiving 40% O_2 (balance N_2) during anesthesia inspired 21% O_2 (balance N_2) during recovery. One turtle inspiring 5% $CO_2/95\%$ O_2 during anesthesia was provided with 100% O_2 during recovery. Second, although all turtles required supportive mechanical ventilation during the recovery period, the method of weaning turtles from mechanical ventilation was varied. Single breaths were provided at intervals of 1 minute ($n = 1$), 5 minutes (2), 10 minutes (3), and 15 minutes (1), or 2 breaths were provided at 10-minute intervals (1). In 6 other turtles, the ventilation rate was gradually reduced by providing 2 successive breaths/min for 15 minutes, followed by 2 breaths once every 4 minutes for 16 minutes and then 2 breaths once every 30 minutes until the turtle was extubated or awake.

Finally, chemical agents were used in 6 turtles to evaluate their effect on time to recovery. These drugs were chosen on the basis of their theoretical ability to increase pulmonary blood flow and, therefore, increase delivery of the anesthetic to the lungs for removal. Five turtles were given atropine sulfate by various routes of administration: 0.04 mg/kg, IM; 0.04 mg/kg, IP; or incremental doses of 0.01 to 0.02 mg/kg administered slowly intra-arterially. All agents administered IP were diluted in 10 ml of saline (0.09% NaCl) solution prior to administration. One turtle was given dobutamine hydrochloride^k at an initial infusion rate of 2 $\mu g/kg/min$, intra-arterially, which was gradually increased to 10 $\mu g/kg/min$. After 23 minutes, atropine (0.04 mg/kg, IP) was administered simultaneously with the highest dobutamine concentration. Dobutamine was discontinued after an

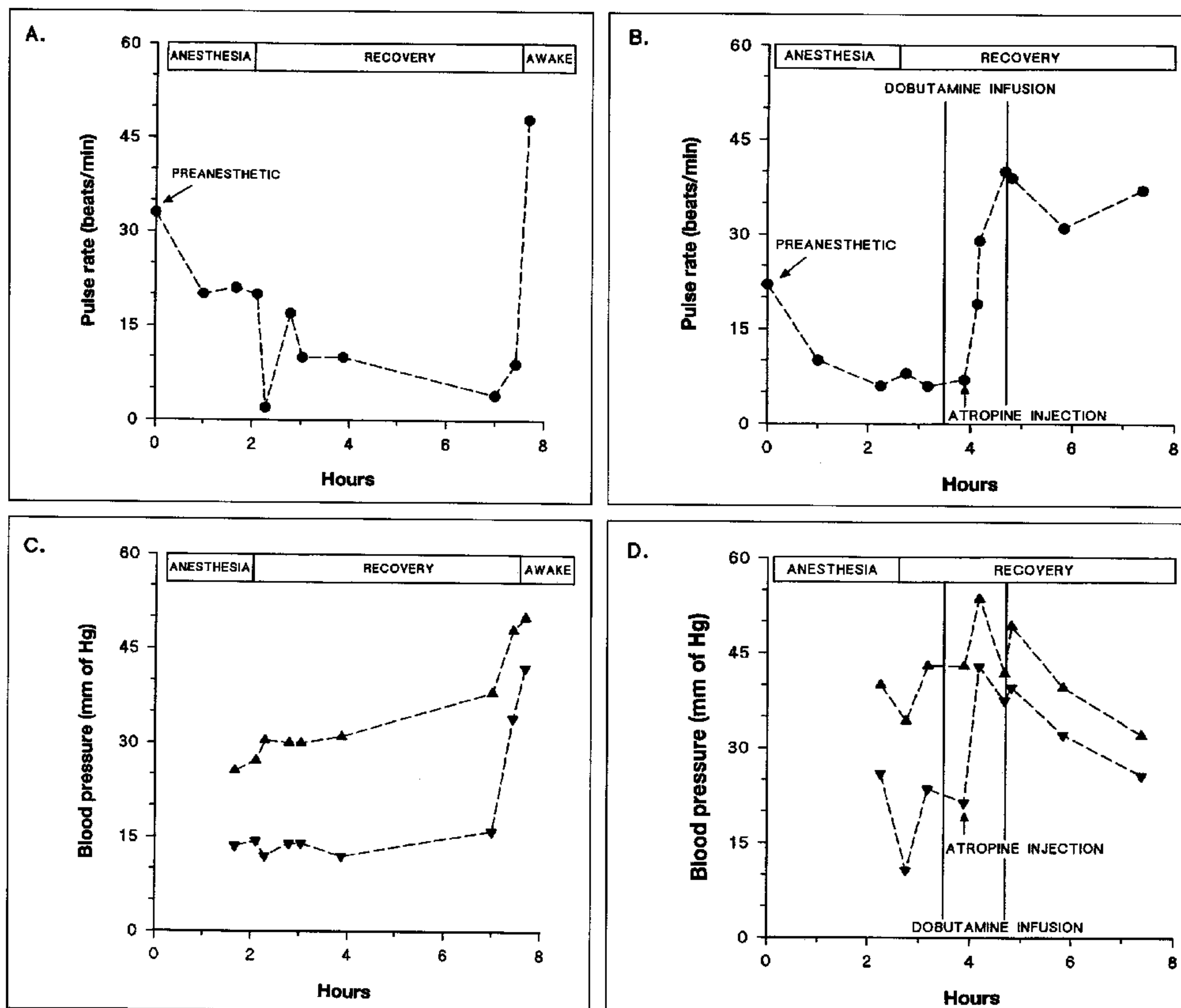


Figure 1—Pulse rate (●) and systolic (▲) and diastolic (▼) systemic blood pressures in the Kemp's ridley sea turtle. Panels A and C are representative traces from a turtle that did not receive pharmacologic agents, whereas panels B and D are representative traces from another turtle that received dobutamine infusion and atropine injection. Pulse rate and blood pressure were measured during the preanesthetic, anesthetic, recovery, and awake periods.

additional 49 minutes of infusion. Naloxone hydrochloride (0.005 ml/kg, IP) was given to 1 turtle as a potential endorphin antagonist to promote CNS stimulation.

Turtles were extubated when they would no longer tolerate the endotracheal tube or when they were awake. Turtles were defined as awake by evidence of concomitant increases in pulse rate and blood pressure and initiation of spontaneous hyperventilation. All turtles were returned to the aquatic housing facility when awake. Throughout the study, the duration of induction (time from induction until dorsal recumbency), surgery, anesthesia, and recovery (time from termination of anesthetic gas until awake) were recorded. All data were calculated as mean \pm SE.

Results

Fourteen anesthetic procedures were conducted on 9 Kemp's ridley sea turtles. All 9 turtles were anesthetized for occlusive carotid artery cannulation, and 5 turtles were reanesthetized 7 to 10 days later for catheter removal. The turtles were 3.7 ± 0.3 years old

and weighed 17 ± 2 kg. The preoperative pulse rate, respiratory rate, and cloacal temperature averaged 34 ± 3 beats/min, 2 ± 1 breaths/min, and 25 ± 3 C, respectively (Table 1).

An isoflurane concentration of $3.4 \pm 0.3\%$ provided anesthetic induction in 7 ± 1 minutes (Table 1). The duration of anesthesia and surgery was 131 ± 12 minutes (range, 75 to 230 minutes) and 102 ± 8 minutes (range, 54 to 158 minutes), respectively. Isoflurane anesthesia induced substantial bradycardia in the sea turtles during the intraoperative period (time from induction to termination of isoflurane). A 56% decrement in pulse rate was measured from the preoperative period to the intraoperative period (Table 1). This bradycardia developed over several minutes and then remained stable throughout the intraoperative period.

The mean duration of the recovery period was 241 ± 31 minutes (range, 92 to 452 minutes). This phase

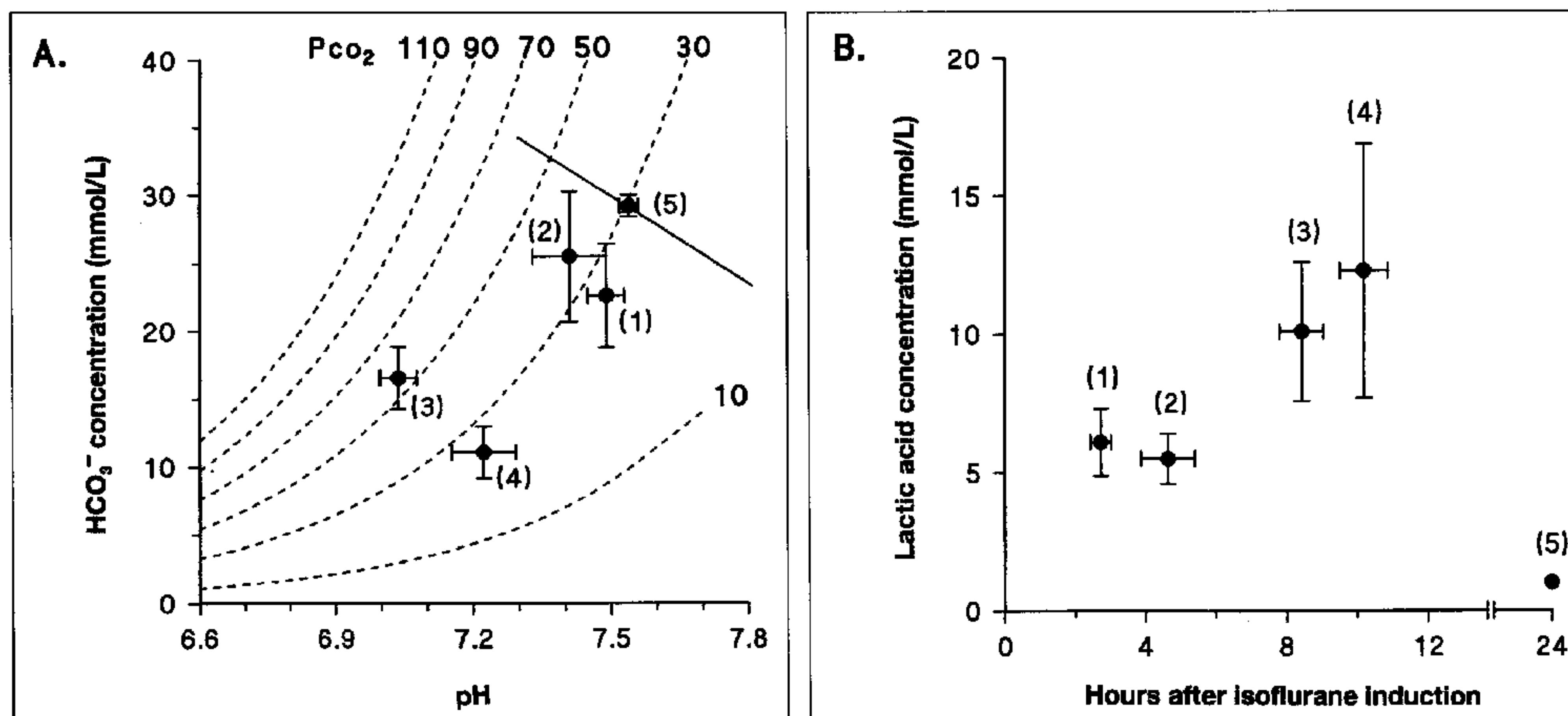


Figure 2—The effects of anesthesia on blood pH, PCO_2 , and HCO_3^- and lactic acid concentrations in Kemp's ridley sea turtles. Panel A—bicarbonate-pH diagram depicting the acid-base changes in the blood of Kemp's ridley sea turtles from the intraoperative (point 1), early and late recovery (points 2 and 3, respectively), and awake period (point 4). Point 5 represents the acid-base status approximately 18 hours after awakening (mean \pm SE; $n = 3$). The dashed lines are PCO_2 isopleths, and the solid line passing through the control value (point 5) is the blood buffer curve. Panel B—concentration of lactic acid in arterial blood measured during the same time periods described in panel A (mean \pm SE; $n = 3$).

exceeded 1.5 hours in all turtles, despite varying the concentration of inspired gases or the method of weaning from mechanical ventilation. Mean pulse rate during the recovery period was identical to that measured during the intraoperative period, although SBp and DBp increased 26% (Table 1).

Although this study was not specifically designed with a control group and an experimental group, the mean duration of the recovery phase was 240 ± 42 minutes (range, 92 to 462 minutes; $n = 8$) for turtles that did not receive drugs and 244 ± 52 minutes (range, 110 to 405 minutes; 6) for turtles that did receive pharmacologic agents (Fig 1). In all turtles that did not receive drugs, a decrement in pulse rate was observed throughout the anesthetic and recovery periods, whereas a substantial increase in pulse rate was measured in the awake turtles (Fig 1A and 1C). Unlike the response exhibited by most mammals, pulse rate and blood pressure did not change in a turtle receiving dobutamine (Fig 1B and 1D). However, substantial increases in pulse rate and blood pressure were measured within 17 minutes of an IP injection of atropine (Fig 1B and 1D). Comparable elevations in pulse rate and blood pressure were measured in all turtles that were given atropine.

Five arterial blood samples were collected from 3 turtles in this study. The first sample was obtained during surgery as soon as the carotid cannulation procedure was completed. The mean time from anesthetic induction to collection of the first blood sample was 2.7 hours. Despite ventilating these turtles with 40% O_2 , the intraoperative PaO_2 was 35 ± 2 mm of Hg (range, 31 to 39 mm of Hg), and PaCO_2 was 26 ± 3 mm of Hg (Fig 2). This period of hypoxia and normocapnia was associated with a profound metabolic

acidosis as indicated on the in vitro buffer curve by a decrease in arterial pH along the 30 mm of Hg PCO_2 isopleth (Fig 2A) and the measurement of high lactic acid concentration (Fig 2B). The second blood gas sample was collected approximately 1.7 hours into the recovery period. The metabolic acidosis had not changed appreciably (Fig 2B), although the PaCO_2 increased 38% and pH decreased 0.08 U (Fig 2A). During the early phase of the recovery period, there was a slight increase in PaO_2 in 2 of the 3 turtles (46 ± 9 mm of Hg; range, 29 to 60 mm of Hg). The third blood gas sample was collected approximately 5.3 hours into the recovery phase. At this time, there was a further increase in lactic acid concentration and PaCO_2 , inducing an additional 0.37 U decrement in arterial pH (Fig 2A and 2B). A 36% decrement in PaO_2 to 29 ± 6 mm of Hg (range, 23 to 36 mm of Hg) also was detected at this time.

The awake state was stereotypic for all turtles. There was a concomitant abrupt onset of spontaneous hyperventilation and tachycardia. Pulse rate increased to values 47, 230, and 230% higher than observed during the preoperative, intraoperative, or recovery periods, respectively (Table 1). In addition, SBp and DBp increased 13 and 60%, respectively, from the recovery period to the awake period (Table 1). The fourth blood gas sample from the 3 turtles was obtained within 3 to 10 minutes of awakening. The PaCO_2 of the awake turtles decreased to 25 ± 4 mm of Hg (Fig 2A), although the lactic acid concentration increased to values greater than those measured during the recovery phase (Fig 2B). The pH increased from 7.04 ± 0.04 during late recovery to 7.22 ± 0.07 in awake turtles. In addition, PaO_2 increased nearly fourfold, from 29 ± 6 mm of Hg during late recovery to 109 ± 5 mm of

Hg (range, 99 to 117 mm of Hg) in awake turtles. The day after anesthesia, the fifth blood gas sample was collected from these turtles. The pH, PaCO₂, and lactic acid concentration (Fig 2A and 2B) had returned to the reference ranges,¹¹ and the PaO₂ was 86 ± 6 mm of Hg (range, 74 to 100 mm of Hg).

The usefulness of capnometry in predicting the PaCO₂ was assessed in 3 turtles during the intraoperative phase and early and late recovery phases by comparing simultaneous measurements of PaCO₂ and end-tidal CO₂ (EtCO₂). During the intraoperative phase (ie, 2.7 hours after anesthetic induction), PaCO₂ was 26 ± 3 mm of Hg, and EtCO₂ was 14 ± 4 mm of Hg; however, PaCO₂ (36 ± 1 mm of Hg) was approximately equal to EtCO₂ (37 ± 3 mm of Hg) during the early phase of the recovery period (ie, 4.4 hours after anesthetic induction). By the late phase of the recovery period (ie, 8.4 hours after anesthetic induction), PaCO₂ and EtCO₂ were 56 ± 3 mm of Hg and 44 ± 2 mm of Hg, respectively.

Discussion

Anesthetic induction of Kemp's ridley sea turtles with isoflurane was rapid, requiring 7 ± 1 minutes. The recovery time ranged from 1.5 to 7.5 hours, despite altering the inspired gas concentration, altering the method of weaning from mechanical ventilation, or the use of selected pharmacologic agents. For comparison, green sea turtles anesthetized with 4% isoflurane required 20 to 50 minutes for mask induction and 2 to 6 hours for recovery from the anesthesia.⁵ These data suggest that our technique of awake intubation and forced manual ventilation of the anesthetic greatly reduced the induction time. Other studies have reported that inhalant anesthetic agents induce prolonged recoveries in reptiles. For example, Custer and Bush¹² reported that gopher snakes required up to 18 hours to recover from halothane anesthesia. In contrast, Shetland ponies recovered to a sustained standing position < 45 minutes after termination of a 2-hour anesthetic period with isoflurane and halothane.¹³ The reasons for the difference in the recovery times between reptiles and mammals is unclear, but there are several plausible explanations.

The prolonged recoveries in the Kemp's ridley sea turtles were most likely caused by intracardiac shunting, intrapulmonary shunting, or ventilation-perfusion (V/Q) mismatch. Although reptiles possess a single ventricle, blood returning to the ventricle from the left and right atria can be directionally ejected into the aorta and the pulmonary vessels with surprisingly little mixing.¹⁴ In addition, many reptile species are capable of altering the amount of blood entering the lungs by altering pulmonary vascular resistance, whereby blood shunts from right to left when pulmonary resistance is increased and blood shunts from left to right when pulmonary resistance is decreased. This mechanism is advantageous in diving turtles, because the animal can increase pulmonary blood flow during air breathing episodes. Thus, factors that increase right-to-left shunting would prolong induction and recovery phases in Kemp's ridley sea turtles. The arterial isoflurane concentration may be diluted as a result of blood bypassing the pulmonary system during anesthetic uptake and, there-

fore, prolong the induction time. Individual variations in the degree of intracardiac shunting during anesthesia also may account for the wide range of isoflurane concentrations required to maintain a surgical plane of anesthesia. During recovery, increased right-to-left shunting would decrease the amount of anesthetic presented to the lungs for removal and would prolong the duration of recovery.

The explanation that V/Q mismatch or intracardiac shunting accounts for the prolonged recoveries in the turtles of this study was supported by experimental evidence. The mean intraoperative PaO₂ was 35 mm of Hg during inspiration of 40% O₂, but the theoretical PaO₂ should have been approximately 200 mm of Hg. This large alveolar-arterial O₂ gradient was measured during normocapnia and was indicative of V/Q mismatch or shunt, rather than hypoventilation. In addition, the effects of positive pressure ventilation may have augmented normal reptilian shunt mechanisms by decreasing venous return and cardiac output through compression of systemic and pulmonary vessels. The cardiovascular effects of 15 cm of H₂O positive pressure ventilation might be greater in reptiles than in mammals because of the low systemic blood pressures and intrinsic intracardiac shunt mechanisms in reptiles. The turtles in this study were also in dorsal recumbency during the intraoperative phase. Compression of the lungs and systemic vessels by the viscera may have enhanced V/Q mismatch or intracardiac shunting. Finally, the depressant effects of general anesthesia may have contributed to the V/Q mismatch or intracardiac shunting by decreasing myocardial contractility and impairing compensatory reflexes. Although the FIO₂ was decreased to 0.21 during recovery, there was an initial slight increase in PaO₂ in 2 of the 3 turtles sampled for blood gas and pH analyses. Therefore, the depressant effects of the anesthetic may have been decreasing and, perhaps, changing the turtles position from dorsal-to-ventral recumbency permitted reexpansion of the pulmonary vasculature. Supportive evidence that there was less V/Q mismatch or intracardiac shunting early in the recovery phase was the comparability of end-tidal and arterial PCO₂. The onset of spontaneous ventilation appeared to improve pulmonary blood flow and minimize right-to-left intracardiac shunting, as the measured PaO₂ was comparable with the theoretical PaO₂ during this time. We conclude that the cardiopulmonary effects of the surgical positioning, anesthesia, and mechanical ventilation were primarily responsible for the prolonged recovery times in the turtles of this study.

We theorized that V/Q mismatch or intracardiac shunting were the most likely causes of the prolonged recoveries in the sea turtles. Lillywhite and Donald¹⁵ reported that pulmonary blood flow in the aquatic file snake (*Acrochordus granulatus*) was controlled by an interaction of adrenergic vasodilation and cholinergic constriction. Atropine abolished the pulmonary vasoconstriction and substantially increased pulmonary blood flow in their study. We hypothesized that increasing pulmonary blood flow by decreasing pulmonary resistance in the sea turtles would permit more anesthetic to be eliminated and, therefore, shorten the

recovery time. Under this assumption, atropine with and without dobutamine was administered to some of these turtles. It is unknown whether atropine or dobutamine administration altered pulmonary blood flow in these turtles, although administration of atropine was consistently associated with increased pulse rate and blood pressure. Neither agent had an apparent effect on the duration of recovery, as evident by the comparable duration of recovery for all turtles in the study. It should be mentioned that the small number of available turtles prevented conducting a controlled study on the physiologic effects of these drugs on recovery time. Nevertheless, the absence of an observable effect on the recovery times suggested that these agents at the doses administered did not decrease the duration of recovery in Kemp's ridley sea turtles.

In addition to these mechanisms, there are 2 other additional factors that may have contributed to the prolonged recovery times. First, the blood/gas and tissue/gas partition coefficients (solubility) of isoflurane may be higher in reptiles than in mammals because of reptiles' lower body temperature¹⁶ and because of differences in lipid and protein composition of reptile blood and tissues.^{17,18} Second, although inhalation anesthetics are primarily eliminated by ventilation in mammals, there are no data to confirm that isoflurane is exclusively eliminated by ventilation in reptiles; therefore, increases in anesthetic solubility and metabolism may influence the recovery phase.

The onset of spontaneous ventilation was associated with general awakening in Kemp's ridley sea turtles. The normal breathing pattern in turtles consists of a series of breaths followed by an end-inspiratory pause of variable duration.¹⁹ Turtles possess various receptors that may be responsible for terminating this nonventilatory period, including pulmonary stretch receptors²⁰ and central²¹ and peripheral chemoreceptors.²² The most important stimulus that triggers breathing in undisturbed snapping turtles (*Chelydra serpentina*) is an increase in PaCO_2 or the concomitant decrease in arterial pH.²³ We used an inspired gas concentration of 5% CO_2 /95% O_2 during the intraoperative period in 1 turtle to determine whether the increased blood CO_2 would reduce the duration of recovery. Blood gas results indicated that this turtle developed a severe respiratory acidosis (eg, $\text{PaCO}_2 > 90$ mm of Hg); therefore, this inspired gas concentration was not used with additional turtles. It is noteworthy that the 92-minute recovery phase for this turtle was the lowest in the study, despite being anesthetized for the longest duration (230 minutes).

Severe hypoxia, hypercapnia, and acidemia were measured during the late recovery phase from 3 turtles. In addition, a metabolic acidosis developed intraoperatively, progressed during recovery, and reached a maximum value upon awakening. This metabolic acidosis, indicative of anaerobic metabolism, was presumably caused by a combination of poor arterial oxygen saturation and poor oxygen delivery to the tissues. Thus, the respiratory and metabolic acidosis combined to induce a clinically profound acidemia. The hypoxia and hypercapnia resolved within 10 minutes of awakening as a result of a fourfold increase in ventilation

from the normal preanesthetized respiratory rate and, presumably, from an improvement in \dot{V}/\dot{Q} matching. A comparable hyperventilation was observed in *Chrysemys scripta* after 2 to 4 hours of diving, despite maintaining normal blood gases and pH.²⁴ A separate study demonstrated that the hyperventilation in turtles recovering from anoxia may be caused by a metabolic acidosis decreasing the pH of the cerebral extracellular fluid, thereby stimulating central chemoreceptors.²⁵ In the study reported here, the concentration of lactic acid was 12 ± 5 mmol/L at the end of the recovery period. The increase in the concentration in awake turtles suggested that restoration of perfusion induced lactic acid washout from previously underperfused areas. It is possible that a combination of stimulation of central and peripheral chemoreceptors, as well as decreasing isoflurane concentrations, were responsible for the abrupt termination of apnea during the recovery period.

The recovery from anesthesia was stereotypic for all turtles in this study and, perhaps, was comparable to their normal dive reflex. Peripheral blood circulation decreases in diving animals because of increased systemic vascular resistance that causes a reflex bradycardia. Upon surfacing from a dive, increases in heart rate are associated with a redistribution of a substantial portion of the blood volume through the pulmonary vasculature, which permits rapid blood oxygenation and carbon dioxide removal. All turtles in the study reported here became bradycardiac and apneic within minutes of isoflurane anesthesia. Pulse rates of 1 to 2 beats/min were recorded from several turtles. Although the specific mechanism is unclear, this bradycardia was vagally mediated based on the responsiveness to atropine and, therefore, was similar to the dive reflex bradycardia. Shaw et al⁵ used a mask induction technique during isoflurane anesthesia and did not report a bradycardia in the green sea turtle. Therefore, it is plausible that the bradycardia and apnea identified in the turtles of this study may have been a reflex response to placement of the endotracheal tube in an awake turtle and not because of the anesthetic.

In the study reported here, the most rapid and reliable technique for pulse rate measurement was obtained by placing the Doppler crystal over the femoral triangle. Placing the crystal over alternative sites produced variable and inconsistent results. Similarly, consistent heart rate measurements could not be obtained with an esophageal stethoscope, lead-II electrocardiography, or pulse oximetry. The Doppler crystal, therefore, was an essential monitoring tool during anesthesia of the Kemp's ridley sea turtles. In contrast, estimating the adequacy of ventilation with capnography had spurious results. Simultaneous measurement of PaCO_2 and EtCO_2 indicated that there was a larger end-tidal to arterial CO_2 gradient than originally assumed and that this gradient would change unpredictably during an experiment with a single animal. These data suggest that capnography should not be used for determining the adequacy of ventilation in sea turtles.

Statistically valid group comparisons were not possible in this study, because we attempted various manipulations with a small number of available turtles

to reduce the length of the recovery phase. However, the mechanisms responsible for the severe acid-base disturbances and the prolonged recovery duration (eg, cardiopulmonary shunts) require further investigation.

^aAerrane, Anaquest, Madison, Wis.

^bModel AO-1, Puritan-Bennett, Los Angeles, Calif.

^cRascal 1, Ohmeda, Madison, Wis.

^dOhio anesthesia ventilator, Ohio Medical Supplies, Madison, Wis.

^eParks Medical Electronics Inc, Cherry Hill, NJ.

^fModel 503, Criticare Systems Inc, Waukesha, Wis.

^gYSI Co Inc, Yellow Springs, Ohio.

^hModel 35-V8404-10, Gould Electronics Recording Systems Division, Cleveland, Ohio.

ⁱpH Electrode Type G297/G2, PO₂ Electrode Type E5046, and PCO₂ Electrode type E5036, Radiometer, Copenhagen, Denmark.

^jKit 826-UV, Sigma Chemical Co, St Louis, Mo.

^kDobutrex, Eli Lilly Co, Carolina, Puerto Rico.

^lAstra Pharmaceutical Products Inc, Westborough, Mass.

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